In The Specification

Please amend the specification as follows:

At page 1, line 7, delete "one or more grants" and insert --National Institutes of Health RO1 40416 and CA34183--.

In the Claims

Please cancel claims 45-47 and amend the claims as follows (all of the pending claims, whether or not amended, are set forth below for the Examiner's convenience):

48. (Amended) A method for modulating [unresponsiveness by a] T cell responsiveness, comprising contacting a T cell which expresses a cytokine receptor γ chain with an antibody which binds to and transduces a signal via the γ chain such that T cell responsiveness is modulated or (i) contacting a T cell which expresses a cytokine receptor γ chain [and has received a primary activation signal] with an agent which modulates a signal associated with ligation of the cytokine receptor γ chain such that [unresponsiveness by the] T cell responsiveness is modulated, [with the proviso that the agent does not consist of natural interleukin-2] and (ii) detecting whether signal transduction via the cytokine receptor γ chain occurs.

49. (Amended) The method of claim 48, wherein the agent stimulates a signal associated with ligation of the cytokine receptor γ chain, such that [unresponsiveness by the] T cell stimulation occurs [is inhibited].

50. (Amended) The method of claim 49, wherein the T cell has received a primary activation signal [under conditions which normally result in unresponsiveness in a T cell] in the absence of a costimulatory signal.

51. (Amended) The method of claim 50, wherein the agent acts extracellularly to stimulate a signal associated with ligation of the cytokine receptor γ chain such that [unresponsiveness by] the T cell is [inhibited] stimulated.

52. The method of claim 51, wherein the agent interacts with the cytokine receptor γ chain.

53. The method of claim 52, wherein the agent is interleukin-4 or interleukin-7.

54. The method of claim 52, wherein the agent is an anti-γ chain antibody.

55. The method of claim 51, wherein the T cell is contacted *in vivo* with the agent.

56. (Amended) The method of claim [51] 48, further comprising contacting the T cell with both an agent which stimulates a primary activation signal in the T cell [and an agent which stimulates a signal associated with ligation of the cytokine receptor γ chain].

~~57~~. The method of claim ~~56~~, further comprising contacting the T cell with an agent which stimulates a costimulatory signal in the T cell.

~~58~~. The method of claim ~~56~~, wherein the agent which stimulates a primary activation signal in the T cell is an antigen.

~~59~~. (Amended) The method of claim ~~58~~, wherein the antigen is a pathogen or portion thereof selected from the group consisting of a virus, a bacteria, and a parasite.

~~60~~. The method of claim ~~58~~, wherein the antigen is a tumor antigen.

~~61~~. The method of claim ~~58~~, wherein the T cell is contacted with the antigen *in vivo*.

62. (Amended) The method of claim 50, wherein the agent acts intracellularly to stimulate a signal associated with ligation of the cytokine receptor γ chain such that [unresponsiveness by] the T cell is [inhibited] stimulated.

63. (Amended) The method of claim 62, wherein the agent acts intracellularly to stimulate phosphorylation of a JAK kinase having a molecular weight of about 116 kD as determined by sodium dodecyl sulfate polyacrylamide gel electrophoresis, such that [unresponsiveness by] the T cell is [inhibited] stimulated.

64. The method of claim 63, wherein the T cell is contacted *in vivo* with the agent.

65. The method of claim 63, further comprising contacting the T cell with both an agent which stimulates a primary activation signal in the T cell and an agent which acts intracellularly to stimulate phosphorylation of a JAK kinase having a molecular weight of about 116 kD as determined by sodium dodecyl sulfate polyacrylamide gel electrophoresis.

66. The method of claim 65, further comprising contacting the T cell with an agent which stimulates a costimulatory signal in the T cell.

67. The method of claim 65, wherein the agent which stimulates a primary activation signal in the T cell is an antigen.

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68. (Amended) The method of claim 67, wherein the antigen is a pathogen or portion thereof, selected from the group consisting of a virus, a bacteria, and a parasite.

69. The method of claim 67, wherein the antigen is a tumor antigen.

70. The method of claim 67, wherein the T cell is contacted with the antigen *in vivo*.

71. (Amended) The method of claim 48, wherein the agent inhibits a signal associated with ligation of the cytokine receptor γ chain, such that [unresponsiveness by the] T cell [is stimulated] unresponsiveness occurs.

72. The method of claim 71, wherein the agent acts extracellularly to inhibit delivery of a signal associated with the cytokine receptor γ chain.

73. The method of claim 72, wherein the agent binds to the cytokine receptor γ chain without stimulating a signal associated with the cytokine receptor γ chain in the T cell.

74. The method of claim 73, wherein the agent is an anti-γ chain antibody.

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75. The method of claim 72, wherein the agent binds a natural ligand of the cytokine receptor γ chain to inhibit binding of the ligand to the cytokine receptor γ chain.

76. The method of claim 75, wherein the agent is selected from the group consisting of an anti-interleukin-2 antibody, an anti-interleukin-4 antibody and an anti-interleukin-7 antibody.

77. The method of claim 71, wherein the agent acts intracellularly to inhibit a signal associated with the cytokine receptor γ chain.

78. The method of claim 77, wherein the agent inhibits association of the cytokine receptor γ chain with a JAK kinase having a molecular weight of about 116 kD as determined by sodium dodecyl sulfate polyacrylamide gel electrophoresis.

79. The method of claim 77, wherein the agent inhibits tyrosine phosphorylation of a JAK kinase having a molecular weight of about 116 kD as determined by sodium dodecyl sulfate polyacrylamide gel electrophoresis.

80. The method of claim 77, wherein the agent inhibits tyrosine phosphorylation of the cytokine receptor γ chain.

81. The method of claim 77, wherein the agent inhibits tyrosine phosphorylation of both the cytokine receptor γ chain and a JAK kinase having a molecular weight of about 116 kD as determined by sodium dodecyl sulfate polyacrylamide gel electrophoresis.

82. The method of claim 71, wherein the T cell is contacted *in vivo* with the agent.

83. The method of claim 71, wherein the primary activation signal is delivered by an antigen.

84. The method of claim 83, wherein the antigen is an alloantigen.

85. The method of claim 83, wherein the antigen is an autoantigen.

86. The method of claim 83, wherein the T cell is contacted with the antigen and the agent *in vitro* and the method further comprises administering the T cell to a subject.

87. The method of claim 86, wherein the antigen is on a surface of an allogeneic or xenogeneic cell and the subject is a recipient of an allogenic or xenogeneic cell.

88. The method of claim 86, wherein the subject is suffering from an autoimmune disease or disorder associated with an inappropriate or abnormal immune response.

89. The method of claim 71, wherein the T cell is a donor T cell in bone marrow and the primary activation signal is delivered by a cell which expresses a recipient antigen, resulting in donor T cell unresponsiveness to the cell which expresses the recipient antigen and inhibition of graft-versus-host disease in a bone marrow transplant recipient.

90. The method of claim 89, wherein the agent is an anti-γ chain antibody.

91. The method of claim 89, wherein the agent binds a natural ligand of the cytokine receptor γ chain to inhibit binding of the ligand to the cytokine receptor γ chain.

92. The method of claim 91, wherein the agent is selected from the group consisting of an anti-interleukin-2 antibody, an anti-interleukin-4 antibody and an anti-interleukin-7 antibody.

93. The method of claim 91, wherein the agent inhibits association of the cytokine receptor γ chain with a JAK kinase having a molecular weight of about 116 kD as determined by sodium dodecyl sulfate polyacrylamide gel electrophoresis.

94. The method of claim 91, wherein the agent inhibits tyrosine phosphorylation of a JAK kinase having a molecular weight of about 116 kD as determined by sodium dodecyl sulfate polyacrylamide gel electrophoresis.